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Letter to the Editor

Case report: Femoral neuropathy with conduction block

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Dear Editor

Femoral neuropathy with conduction block is uncommon. It has been reported due to stretch injury, compression from hematoma [1], complications from surgery [2], and other lesions [3,4] To our knowledge, there has not been a report of conduction block in the femoral nerve due to drug overdose. We report such a case in an adolescent male.

1. Case report

A 16-year-old high school student in previous good health awoke one morning with painless left thigh weakness and numbness. He initially denied substance abuse, but the emergency department note from the day of onset indicated that he did admit to snorting crushed Xanax the previous night. Following this, he was unconscious for a prolonged but imprecise time period (several hours minimum). CT imaging revealed a 2 cm area of signal attenuation in his left iliopsoas at the level of the inguinal ligament consistent with fluid collection that was thought to be inflammatory. Serum CK was initially 7376 IU at day of onset but decreased to 277 IU six days later. Core needle muscle biopsy was performed of the left psoas distal to the inguinal ligament in an area felt to be congruent with the CT findings which was reported as normal. It was uncertain whether the specimen was obtained from the abnormal area seen on CT scan, thus representing sampling error.

He was seen in the Neuromuscular Clinic ten days after onset. General examination was unremarkable. No masses or pain was evident in the left leg. Strength in the left hip flexors was 4 of 5 and the knee extensors 3 of 5. There was a 2-cm difference between the left and right thigh circumference (45 and 47 cm, respectively). The left knee myotatic stretch response was absent on the left and 2+ on the right. Sensation to sharp touch was decreased in the left anterior thigh and the anteromedial aspect of the left distal leg on clinical examination, consistent with the cutaneous distribution of the femoral nerve. He ambulated with a limp in the left leg. Femoral stretch test elicited no pain. The remainder of the examination was unremarkable. He was referred for an EMG.

Electrodiagnostic study was performed 28 days post-onset (see Fig. 1): The left femoral motor conduction showed normal latency, velocity and distal amplitude recording with surface electrodes from the quadriceps (modified Rigshospsitalet method [5]). Moving the stimulator proximally, the evoked response from above the inguinal ligament showed a 56% drop in amplitude and area (see Fig. 1). Supramaximal stimulus intensity was verified in two ways: increasing it by 25% once a maximal response was obtained and comparing the intensity needed for supramaximal stimulation on the unaffected side).

The right femoral motor conduction study was normal, showing a similar response to that of the right distal response.

Sensory nerve conduction of the right saphenous nerve was normal (3.0 μV) using the Wainapel method of distal saphenous sensory conduction [5]; that of the left was absent. Sensory conductions of both lateral cutaneous nerves of the thigh were normal (right 8.9 μV; left 7.9 μV). Motor conductions in the left fibular and tibial nerves were normal. The sensory conductions in the left sural and superficial fibular nerves were normal. On needle electrode examination no fibrillation potentials or abnormal insertion activity were present. The motor unit potentials in the left vastus medialis, vastus lateralis, and iliacus were normal in appearance with reduced recruitment. The other muscles tested (left tibialis anterior, medial gastrocnemius, adductor longus, lumbar paraspinal) were normal. Interpretation was conduction block of the left femoral nerve.

A second electrodiagnostic study was performed 12 weeks post-onset. His clinical examination showed normal strength in both quadriceps and hip flexors. The area of numbness in his left thigh had resolved compared to his initial examination, with residual, but improving numbness in the left anteromedial distal leg (saphenous nerve distribution). Gait was normal. A limited repeat study showed resolution of the left femoral motor conduction block (see Fig. 1). The amplitudes of the motor responses were slightly higher on the left on the second study, however the differences were minimal and likely related to positioning of the E1 (active) electrode and mild muscle wasting. The left saphenous sensory conduction remained unobtainable consistent with the residual clinical sensory deficit.

One week after the first EMG (5 weeks post symptom onset), he developed acute shortness of breath with pain on inspiration. He was admitted to hospital because of concerns for pulmonary embolism. Spiral chest CT scan demonstrated cavitary lesions in the lateral pleura of the right lung. Biopsy of the lung and pleura showed pigmented inflammatory lesions secondary to the snorted Xanax. This was felt to be secondary to the initial event and supported the history of his

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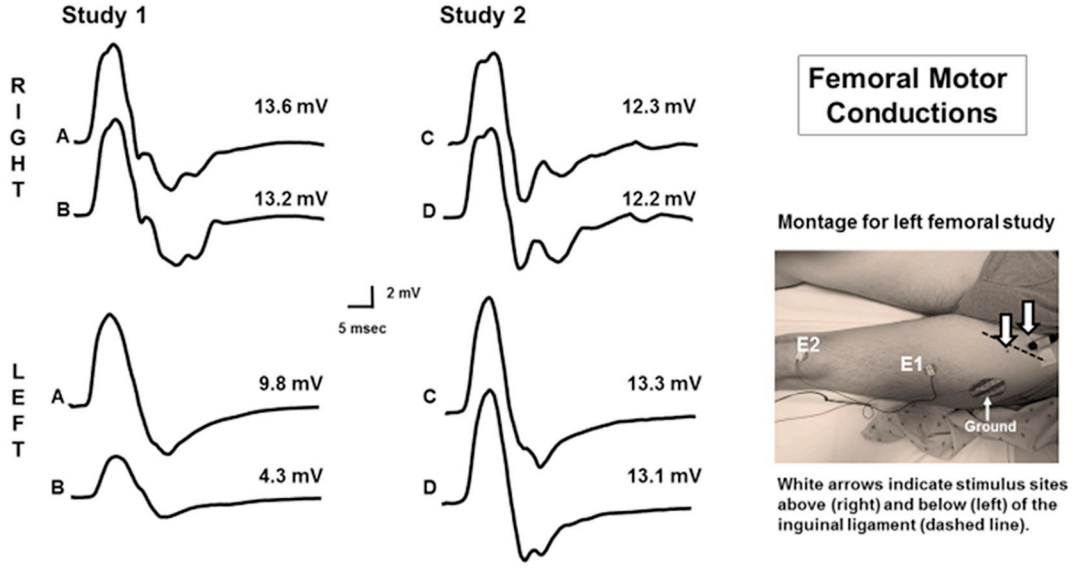

Fig. 1. Femoral motor conductions shown at first and second studies. Unaffected side (right) are top tracings showing minimal difference in amplitude below and above the inguinal ligament and 10% variation between first and second study. On the affected left side, there is a 56% conduction block on the first study that is resolved at the second study. On the second study there is good symmetry in amplitudes of the evoked responses on each side. The picture demonstrates the recording montage on the left. The same montage is used on the right. The clothing on the left is drawn up higher to allow better visualization of the segment stimulated. See text for details.

substance abuse. Repeat chest CT 13 months from presentation showed resolution of cavitary lesions. His gait has normalized with almost complete resolution of the numbness in his distal leg.

2. Discussion

Focal neuropathies of unknown cause and without apparent reason require pursuit of an accurate history. In this case, the patient was not entirely forthcoming regarding his substance abuse, creating initial confusion regarding cause of his neuropathy. As he was deeply self-sedated, the precise mechanism of the lesion will remain unknown; but it caused him to be in a position where his femoral nerve was focally compressed at the inguinal ligament, such as may be seen in extreme hip flexion in obstetric patients in active labor [6]. The fluid accumulation in the underlying iliopsoas muscle on the CT scan and acutely elevated CK further suggest that this was due to prolonged immobility.

The electrodiagnostic studies were helpful in demonstrating that the conduction block in the femoral nerve was due to focal demyelination with good prognosis. This was subsequently confirmed by his clinical course and second electrodiagnostic study. Finally, conduction block can be demonstrated in the femoral nerve as shown in this study. In demonstrating focal conduction block over a short distance (2–3 cm), the examiner would follow the same precautions as in performing "inching studies" used to assess patients for focal ulnar neuropathy at the elbow [7].

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